```
$%^Dialog;HighlightOn=%%%;HighlightOff=%%%;

Connecting via Winsock to Dialog


Logging in to Dialog

Trying 31060000009998...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ********
ENTER PASSWORD:
 ********
Welcome to DIALOG

Dialog level 05.24.00D

Last logoff:  18jun09 10:20:27
Logon file1 18jun09 16:30:45
* * *

File   1:ERIC 1965-2009/May
       (c) format only 2009 Dialog

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
? b 410
       18jun09 16:30:46 User219511 Session D765.1
            $0.55    0.152 DialUnits File1
     $0.55  Estimated cost File1
     $0.55  Estimated cost this search
     $0.55  Estimated total session cost   0.152 DialUnits

File 410:Dialog Customer Newsletters 2008
       (c) 2009 Dialog. All rts. reserv.

      Set  Items  Description
      ---  -----  -----------
? set hi %%%;set hi %%%
HILIGHT set on as '%%%'%%%
%%%HILIGHT set on as '%%%'
? b 411;set files biotech
       18jun09 16:30:52 User219511 Session D765.2
            $0.00    0.117 DialUnits File410
     $0.00  Estimated cost File410
     $0.02  TELNET
     $0.02  Estimated cost this search
     $0.57  Estimated total session cost   0.269 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
   (c) 2009 Dialog

*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
```

```
   You have 27 files in your file list.
   (To see banners, use SHOW FILES command)
? s (aquaporin3 or aquaporin-3) and (immune? or immuno?)

Your SELECT statement is:
   s (aquaporin3 or aquaporin-3) and (immune? or immuno?)

          Items   File
          -----   ----
             50     5: Biosis Previews(R)_1926-2009/Jun W2
             18    34: SciSearch(R) Cited Ref Sci_1990-2009/Jun W2
              4    71: ELSEVIER BIOBASE_1994-2009/Jun W2
              1   154: MEDLINE(R)_1990-2009/Jun 17
              1   155: MEDLINE(R)_1950-2009/Jun 17
              1   172: EMBASE Alert_2009/Jun 17

   6 files have one or more items; file list includes 27 files.

? save temp; b 154,155,5,71;exs;rd
Temp SearchSave "TD689321579" stored
       18jun09 16:32:10 User219511 Session D765.3
            $6.84    2.326 DialUnits File411
     $6.84  Estimated cost File411
     $0.53  TELNET
     $7.37  Estimated cost this search
     $7.94  Estimated total session cost   2.595 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File 154:MEDLINE(R) 1990-2009/Jun 17
         (c) format only 2009 Dialog
  File 155:MEDLINE(R) 1950-2009/Jun 17
         (c) format only 2009 Dialog
  File   5:Biosis Previews(R) 1926-2009/Jun W2
         (c) 2009 The Thomson Corporation
  File  71:ELSEVIER BIOBASE 1994-2009/Jun W2
         (c) 2009 Elsevier B.V.
*File  71: The file has been reloaded. Accession numbers
have changed.

      Set  Items  Description
      ---  -----  -----------
Executing TD689321579
Processing
             5  AQUAPORIN3
           158  AQUAPORIN-3
       2762184  IMMUNE?
       5987319  IMMUNO?
      S1      56  (AQUAPORIN3 OR AQUAPORIN-3) AND (IMMUNE? OR IMMUNO?)
      S2      52  RD  (unique items)
? t s2/7/1-52

 2/7/1     (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

16915847   PMID: 16183643
  Heregulin   induces   glial   cell   line-derived   neurotrophic   growth
```

factor-independent, non-branching growth and differentiation of ureteric bud epithelia.
Sakurai Hiroyuki; Bush Kevin T; Nigam Sanjay K
Division of Nephrology-Hypertension, Department of Medicine, University of California San Diego, La Jolla, California 92093, USA.
Journal of biological chemistry (United States) Dec 23 2005, 280 (51) p42181-7, ISSN 0021-9258--Print Journal Code: 2985121R
Contract/Grant No.: DK57286; DK; NIDDK NIH HHS United States
Publishing Model Print-Electronic
Document type: Journal Article; Research Support, N.I.H., Extramural; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
We have purified a protein present in a conditioned medium derived from the metanephric mesenchyme that supports non-branching growth and epithelial differentiation of the isolated ureteric bud (UB) independent of glial cell line-derived neurotrophic growth factor (GDNF). By sequential liquid chromatography, together with protein microsequencing, the protein was identified as heregulin (HRG)alpha. The addition of recombinant HRG to the isolated UB grown in three-dimensional culture confirmed the proliferative activity of HRG. In branching UBs induced by whole metanephric mesenchyme cell-conditioned medium, proliferating cells were localized at ampullae, where a binding receptor for GDNF, GFRalpha1, was found. In HRG-induced UBs, however, the expression of GFRalpha1 was down-regulated, and proliferating cells were distributed throughout the structure. Electron microscopic examination of the HRG-induced UB revealed the presence of structurally mature and polarized epithelial cells reminiscent of the epithelial cells found in the stalk portion of the branching UB. cDNA array analysis further revealed that genes ontologically classified as developmental were down-regulated by HRG, whereas those involved in transport were up-regulated. For example, the mRNA for the GDNF receptors, GFRalpha1 and ret9, was down-regulated, whereas the mRNA for collecting duct transporters, such as urea transporter2, %%%aquaporin3%%%, and sodium-hydrogen exchanger2 was up-regulated in HRG-treated UBs compared with UBs grown in the presence of branch-promoting factors. Moreover, HRG promoted growth of UBs cultured in the absence of GDNF. Taken together, the data suggest that HRG supports UB epithelial cell differentiation and non-GDNF-dependent growth, raising the possibility that this kind of activity plays a role in the growth and differentiation of the stalk portion of the branching epithelial UB.
Record Date Created: 20051219
Record Date Completed: 20060404
Date of Electronic Publication: 20050923

2/7/2 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020869897 BIOSIS NO.: 200900210231
The expression of differentiation markers in aquaporin-3 deficient epidermis
AUTHOR: Hara-Chikuma Mariko (Reprint); Takahashi Kenzo; Chikuma Shunsuke; Verkman A S; Miyachi Yoshiki
AUTHOR ADDRESS: Kyoto Univ, Dept Dermatol, Grad Sch Med, Sakyo Ku, 54 Kawahara Cho, Kyoto 6068507, Japan**Japan

AUTHOR E-MAIL ADDRESS: haramari@kuhp.kyoto-u.ac.jp
JOURNAL: Archives of Dermatological Research 301 (3): p245-252 MAR 2009 2009
ITEM IDENTIFIER: doi:10.1007/s00403-009-0927-9
ISSN: 0340-3696
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aquaporin-3 (AQP3) is a water/glycerol transporting protein expressed strongly at the plasma membrane of keratinocytes. There is evidence for involvement of AQP3-facilitated water and glycerol transport in keratinocyte migration and proliferation, respectively. Here, we investigated the involvement of AQP3 in keratinocyte differentiation. Studies were done using AQP3 knockout mice, primary cultures of mouse keratinocytes (AQP3 knockout), neonatal human keratinocytes (AQP3 knockdown), and human skin. Cells were cultured with high Ca2+ or 1 alpha,25-dihydroxyvitamin D-3 (VD3) to induce differentiation. The expression of differentiation marker proteins and differentiating responses were comparable in control and AQP3-knockout or knockdown keratinocytes. Topical application of all-trans retinoic acid (RA), a known regulator of keratinocyte differentiation and proliferation, induced comparable expression of differentiation marker proteins in wildtype and AQP3 null epidermis, though with impaired RA-induced proliferation in AQP3 null mice. %%%Immunostaining%%% of human and mouse epidermis showed greater AQP3 expression in cells undergoing proliferation than differentiation. Our results showed little influence of AQP3 on keratinocyte differentiation, and provide further support for the proposed involvement of AQP3-facilitated cell proliferation.

2/7/3 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020290507 BIOSIS NO.: 200800337446
Aquaporin-8 is involved in water transport in isolated superficial colonocytes from rat proximal colon
AUTHOR: Laforenza Umberto (Reprint); Cova Emanuela; Gastaldi Giulia; Tritto Simona; Grazioli Monica; LaRusso Nicholas F; Splinter Patrick L; D'Adamo Patrizia; Tosco Marisa; Ventura Ulderico
AUTHOR ADDRESS: Univ Pavia, Dipartimento Med Sperimentale, Sezione Fisiol Umana, I-27100 Pavia, Italy**Italy
AUTHOR E-MAIL ADDRESS: lumberto@unipv.it
JOURNAL: Journal of Nutrition 135 (10): p2329-2336 OCT 2005 2005
ISSN: 0022-3166
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Water is an essential nutrient because it must be introduced from exogenous sources to satisfy metabolic demand. Under physiologic conditions, the colon can absorb and secrete considerable amounts of water even against osmotic gradients, thus helping to maintain the body fluid balance. Here we describe studies on both aquaporin (AQP) expression and function using cells isolated from the superficial and lower crypt regions of the rat proximal colon. The expression of AQP-3,

-4, and -8 in isolated colonocytes was determined by semiquantitative RT-PCR and by %%%immunoblotting%%%. The localization of AQP-8 in the colon was evaluated by %%%immunohistochemistry%%%. A stopped-flow light scattering method was used to examine osmotic water movement in isolated colonocytes. Moreover, the contribution of AQP-8 to overall water movement through isolated colonocytes was studied using RNA interference technology. Colonocytes from the proximal colon express AQP-3, -4, and -8 with increasing concentrations from the lower crypt cells toward those on the surface. Osmotic water permeability was higher in surface than in crypt colonocytes ($P < 0.05$); it was significantly inhibited by the water channel blocker dimethyl sulfoxide, and reversed by P-mercaptoethanol ($P < 0.05$). %%%Immunohistochemistry%%% revealed a strong AQP-8 labeling in the apical membrane of the superficial colonocytes. Inhibition of aquaporin-8 expression by small interfering RNA significantly decreased osmotic water permeability (similar to 38%; $P < 0.05$). Current results indicate that aquaporin-8 may play a major role in water movement through the colon by acting on the apical side of the superficial cells.

2/7/4 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020226351 BIOSIS NO.: 200800273290
Increased expression of aquaporin-3 in the epidermis of DHCR24 knockout mice
AUTHOR: Mirza R; Hayasaka S; Kambe F; Maki K; Kaji T; Murata Y; Seo H (Reprint)
AUTHOR ADDRESS: Nagoya Univ, Chikusa Ku, Res Inst Environm Med, Dept Endocrinol,Div Stress Recognit and Response, Furo Cho, Nagoya, Aichi 4648601, Japan**Japan
AUTHOR E-MAIL ADDRESS: hseo@riem.nagoya-u.ac.jp
JOURNAL: British Journal of Dermatology 158 (4): p679-684 APR 2008 2008
ITEM IDENTIFIER: doi:10.1111/j.1365-2133.2007.08424.x
ISSN: 0007-0963
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background The DHCR24 (3 beta-hydroxysterol-Delta 24 reductase) gene encodes an enzyme catalysing conversion of desmosterol to cholesterol. Desmosterolosis is an autosomal recessive disease due to mutation in the DHCR24 gene, with low cholesterol and high desmosterol levels. To understand the pathophysiology of this disease, we utilized DHCR24 knockout mice and reported that DHCR24-/- mice die soon after birth. Their skin was less wrinkled, shiny, and revealed features of lethal restrictive dermopathy associated with severe defects in epidermal maturation and barrier function.Objectives Markedly increased transepidermal water loss in DHCR24-/- mice led us to examine the role of aquaporin-3 (AQP3), because this is the only water/glycerol transporting channel protein expressed in the epidermis.Methods Expression of AQP3 was studied by Western blot analysis and %%%immunohistochemistry%%% in the epidermis of DHCR24-/- and wild-type newborn mice. Glycerol uptake was determined in the isolated keratinocytes and glycerol content in the epidermis was analysed by an enzymatic method.Results In control mice, AQP3 was expressed only in cells of the stratum basale, indicating its expression in immature keratinocytes. In DHCR24-/- mice, AQP3 was

expressed throughout the epidermis and colocalized with the immature keratinocytes (keratin 14-positive cells). The increased AQP3 expression in the epidermis of DHCR24-/- mice was mirrored by a significantly higher glycerol uptake and glycerol content. This was associated with an increase in epidermal water content of DHCR24-/- mice.Conclution This is the first demonstration that elevated AQP3 results in the retention of epidermal water, causing the taut, wrinkle-free skin phenotype of the DHCR24-/- mice.

2/7/5 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020214817 BIOSIS NO.: 200800261756
Retinoic acid increases aquaporin 3 expression in normal human skin
AUTHOR: Bellemere Gaelle (Reprint); Von Stetten Otto; Oddos Thierry
AUTHOR ADDRESS: Johnson and Johnson Consumer Prod Inc, Vitro Pharmacotoxicol Lab, Campus Maigremont, F-27100 Val de Reuil, France**France
AUTHOR E-MAIL ADDRESS: gbelleme@jacfr.jnj.com
JOURNAL: Journal of Investigative Dermatology 128 (3): p542-548 MAR 2008 2008
ITEM IDENTIFIER: doi:10.1038/sj.jid.5701047
ISSN: 0022-202X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have investigated the effects of all-trans retinoic acid (ATRA) on aquaporin 3 (AQP3) expression and function both in vitro and ex vivo. ATRA treatment provoked a rapid accumulation of AQP3 transcripts in cultured normal human epidermal keratinocytes (NHEK). This increase was still observed 24 hours after application of ATRA. The induction of AQP3 gene was accompanied by an augmentation of %%%immunoreactivity%%%. Using a selective agonist, we demonstrated that the effect of ATRA was predominantly mediated by retinoic acid receptor subtype gamma (PAR gamma). Incubation of NHEK in ATRA for 24, 48, and 72 hours stimulated glycerol influx, suggesting that the increase in AQP3 gene and protein expression was followed by an enhancement of biological activity. Topical application of ATRA for 24 hours on skin explants induced significant epidermal expression of AQP3 and strong %%%immunoreactivity%%% in the epidermal basal layers. Collectively, the present results show that ATRA increased AQP3 expression and enhanced biological activity in human skin.

2/7/6 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019908290 BIOSIS NO.: 200700568031
The role of aquaporin 3 in teleost fish
AUTHOR: Cutler Christopher Paul (Reprint); Martinez Anne-Sophie; Cramb Gordon
AUTHOR ADDRESS: Georgia So Univ, Dept Biol, Statesboro, GA 30460 USA**USA
AUTHOR E-MAIL ADDRESS: ccutler@georgiasouthern.edu
JOURNAL: Comparative Biochemistry and Physiology Part A Molecular &

Integrative Physiology 148 (1): p82-91 SEP 2007 2007
ITEM IDENTIFIER: doi:10.1016/j.cbpa.2006.09.022
ISSN: 1095-6433
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The aquaporin isoform, AQP3 has now been identified in a number of different teleost fish species, with additional DNA sequence information on AQP3 genes in further fish species available in genome databases. In zebrafish (Danio rerio), the AQP3 gene is present as two duplicate isoforms resulting from a teleostean fish genome-wide duplication. A further splicoform/isoform has also been identified in rainbow trout (Oncorhynchus mykiss). The identification of these AQP3 isoforms in other fish species is consequently explored. The role of AQP3 in physiological/osmoregulatory processes, in various teleost organs is then described. In teleost gill, AQP3 is expressed in 'chloride' cells, and in some species, in other epithelial cell types, where it may have a number of different functions including the prevention of dehydration. In eel esophagus, %%%immunohistochemistry%%% shows that AQP3 is expressed in surface epithelial cells in the anterior esophagus, but in mucus cells within the epithelium of the posterior esophagus. In eel intestine, AQP3 is found in macrophage-like cells and probably plays no part in osmoregulatory processes. In the rectum, as in the posterior esophagus AQP3 is expressed in mucus cells. In eel kidney, AQP3 is expressed in a subset of renal tubules, and localizes to the apical pole of tubule cells. There is no apparent change in the location or protein abundance of renal AQP3 following the acclimation of eels from freshwater to seawater. P (c) 2006 Elsevier Inc. All rights reserved.

2/7/7 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019683841 BIOSIS NO.: 200700343582
DHCR24-/- mice demonstrate impaired maturation of the epidermis with an increased expression of aquaporin-3 (AQP3), affecting the epidermal hydration
AUTHOR: Mirza R (Reprint); Hayasaka S; Kambe F; Maki K; Kaji T; Murata Y; Seo H
AUTHOR ADDRESS: Nagoya Univ, Environm Med Res Inst, Dept Endocrinol, Nagoya, Aichi 464, Japan**Japan
JOURNAL: Journal of Investigative Dermatology 127 (Suppl. 1): pS65 APR 2007 2007
CONFERENCE/MEETING: 68th Annual Meeting of the Society-for-Investigative-Dermatology Los Angeles, CA, USA May 09 -12, 2007; 20070509
SPONSOR: Soc Investigat Dermatol
ISSN: 0022-202X
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/8 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019547432 BIOSIS NO.: 200700207173
Increased renal expression of aquaporin-3 in rats inhibited type 2 11 beta-hydroxysteroid dehydrogenase
AUTHOR: Ma Seong Kwon; Nam Kwang Il; Kim Soo Wan; Bae Eun Hui; Choi Ki Chul ; Lee JongUn (Reprint)
AUTHOR ADDRESS: Chonnam Natl Univ, Sch Med, Dept Physiol, 5 Hak Dong, Kwangju 501746, South Korea**South Korea
AUTHOR E-MAIL ADDRESS: julee@jnu.ac.kr
JOURNAL: Kidney & Blood Pressure Research 30 (1): p8-14 2007 2007
ISSN: 1420-4096
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aims: To investigate whether the regulation of aquaporin (AQP) channels is altered by inhibition of type 2 11 beta-hydroxysteroid dehydrogenase (11 beta HSD2). Methods: Male Sprague-Dawley rats were treated with glycyrrhizic acid (GA, 2 g/l drinking water) for 7 days. The expression of AQP2 and AQP3 was determined in the kidney by %%%immunoblotting%%% and %%%immunohistochemistry%%%. The expression of Gs alpha and type VI adenylyl cyclase, and the activity of adenylyl cyclase were also determined. Results: Following the GA treatment, the expression of 11 beta HSD2 was significantly decreased in the kidney. The expression of AQP3 was increased, while that of AQP2 remained unchanged. Plasma renin activity and serum aldosterone levels were decreased. Plasma arginine vasopressin (AVP) levels were comparable between the groups. Neither the forskolin-stimulated cAMP generation nor the expression of Gs alpha and type VI adenylyl cyclase was altered significantly. Conclusion: A decreased expression of 11 beta HSD2 may result in an upregulation of AQP3, in which AVP/cAMP-dependent mechanisms are unlikely to be involved. Copyright (c) 2007 S. Karger AG, Basel.

2/7/9 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019471343 BIOSIS NO.: 200700131084
Phosphatidylglycerol normalizes keratinocyte proliferation in vitro and accelerates wound healing in vivo
AUTHOR: Bollag W B (Reprint); Zhong X
AUTHOR ADDRESS: Med Coll Georgia, Inst Mol Med and Genet, Augusta, GA 30912 USA**USA
JOURNAL: Journal of Investigative Dermatology 126 (Suppl. 1): p94 APR 2006 2006
CONFERENCE/MEETING: 67th Annual Meeting of the Society-for-Investigative-Dermatology Philadelphia, PA, USA May 03 -06, 2006; 20060503
SPONSOR: Soc Investigat Dermatol
ISSN: 0022-202X
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/10 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18974327 BIOSIS NO.: 200600319722
Roles of aquaporin-3 water channels in volume-regulatory water flow in a human epithelial cell line
AUTHOR: Kida H; Miyoshi T; Manabe K; Takahashi N; Konno T; Ueda S; Chiba T; Shimizu T; Okada T (Reprint); Morishima S
AUTHOR ADDRESS: Natl Inst Physiol Sci, Dept Cell Physiol, Okazaki, Aichi 4448585, Japan**Japan
AUTHOR E-MAIL ADDRESS: okada@nips.ac.jp
JOURNAL: Journal of Membrane Biology 208 (1): p55-64 NOV 2005 2005
ISSN: 0022-2631
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Membrane water transport is an essential event not only in the osmotic cell volume change but also in the Subsequent cell volume regulation. Here we investigated the route of water transport involved in the regulatory volume decrease (RVD) that occurs after osmotic swelling in human epithelial Intestine 407 cells. The diffusion water permeability coefficient (Pd) measured by NMR under isotonic conditions was much smaller than the osmotic water permeability coefficient (pf) measured under an osmotic gradient. Temperature dependence of Pf showed the Arrhenius activation energy (Ea) of a low value (1.6 kcal/mol). These results indicate an involvement of a facilitated diffusion mechanism in osmotic water transport. A mercurial water channel blocker (HgCl2) diminished the Pf value. A non-mercurial sulfhydryl reagent (MMTS) was also effective. These blockers of water channels suppressed the RVD. RT-PCR and %%%immunocytochemistry%%% demonstrated predominant expression of AQP3 water channel in this cell line. Downregulation of AQP3 expression induced by treatment with antisense oligodeoxynucleotides was found to suppress the RVD response. Thus, it is concluded that AQP3 water channels serve as an essential pathway for volume-regulatory water transport in, human epithelial cells.

2/7/11 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18879816 BIOSIS NO.: 200600225211
A putative role of secretin to regulate water homeostasis
AUTHOR: Chow B K C (Reprint); Chu J Y S; Chung S C K
AUTHOR ADDRESS: Univ Hong Kong, Dept Zool, Hong Kong, Hong Kong, Peoples R China**Peoples R China
JOURNAL: Regulatory Peptides 130 (3): p137 SEP 15 2005 2005
CONFERENCE/MEETING: 7th International Symposium on VIP, PACAP and Related Peptides Rouen, FRANCE September 11 -14, 2005; 20050911
SPONSOR: Conseil Reg Haute-Normandie
Agglomerat Rouen
Inst Fed Rech Multidisciplinaires Peptides
Inst Natl Sante Rech Med
Municipal Rouen
Sci Act Haute-Normandie

Tech Chime-Biol Sante
Univ Paris 7
Univ Rouen
ISSN: 0167-0115
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

18710064 BIOSIS NO.: 200600055459
Delayed corneal epithelial wound healing in aquaporin-3-null mice
AUTHOR: Levin M H (Reprint); Verkman A S
JOURNAL: IOVS 46 (Suppl. S): p3618 2005 2005
CONFERENCE/MEETING: Annual Meeting of the
Association-for-Research-in-Vision-and-Ophthalmology Ft Lauderdale, FL, USA May 01 -05, 2005; 20050501
SPONSOR: Assoc Res Vis & Ophthalmol
ISSN: 0146-0404
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose: The water/glycerol-transporting protein aquaporin (AQP)-3 is expressed in corneal and conjunctival epithelia in human and mouse eye. Here, we test the hypothesis that AQP3 is involved in corneal epithelial wound healing. The motivation for this study was the previously demonstrated role of AQP3 in epidermal formation after injury, and a recently discovered role of aquaporins in cell migration. Methods: Central corneal epithelial defects were created in wild-type and AQP3-null mice in a CD1 genetic background. Wounds were demarcatedwith a trephine and full-thickness epithelia were removed with a blunt blade without basement membrane disruption. Centripetal advance of epithelia during healing was monitored at specified times by fluorescein imaging. Paraffin sections were also obtained at baseline and at different times after wound creation for morphology and %%%immunohistochemistry%%%. Wild-type AQP3 transcript expression in various ocular surface regions was analyzed by quantitative RT-PCR. In vivo ocular surface open-circuit potential differences (PDs) were recorded to assess knock-out epithelial resistance and ion transport properties. Glycerol content of homogenized full-thickness corneal epithelium was measured by a glycerol oxidase-based colorimetric assay. Results: Baseline corneal morphology andocular PDs were similar in wildtype and AQP3-null mice, suggesting similar corneal epithelial integrity in the two groups. Corneal epithelial glycerol content was not affected by AQP3 deletion (in nmol/mu g protein: 2.0 +/- 0.2 versus 1.7 +/- 0.1 in wildtype mice). %%%Immunohistochemistry%%% and RT-PCR showed strongAQP3 expression in conjunctival epithelium and at the corneal limbus, with relatively weaker expression in the central corneal epithelium. At 24 hours after wound creation, AQP3-null defects were 85 +/- 4% of their original area (versus 33 +/- 1% for wild- type; SE, 8 mice per group). At 48 hours, wounds in most AQP3-deficient mice remained incompletely closed (17 +/- 4% area) compared to wild- type (2 +/- 2%). Histology confirmed that corneas from wildtype mice largely resurfaced with stratified epithelia by 48 hours,

whereas many AQP3-deficient corneas lacked even a basal epithelial cell layer and had significant inflammatory reaction in the anterior stroma. Conclusions: Our results provideevidence for a novel role for AQP3 in corneal wound healing. This defect is probably not related to baseline differences in glycerol content or to the glycerol-transporting role of AQP3. We propose that reduced water permeability in AQP3-null limbal epithelial cells reduces cell migration during healing.

2/7/13 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18559769 BIOSIS NO.: 200510254269
Aquaporin-3 expressed in the basolateral membrane of gill chloride cells in Mozambique tilapia Oreochromis mossambicus adapted to freshwater and seawater
AUTHOR: Watanabe Soichi (Reprint); Kaneko Toyoji; Aida Katsumi
AUTHOR ADDRESS: Univ Tokyo, Grad Sch Agr and Life Sci, Dept Aquat Biosci, Bunkyo Ku, Tokyo 1138657, Japan**Japan
AUTHOR E-MAIL ADDRESS: watanabe@marine.fs.a.u-tokyo.ac.jp
JOURNAL: Journal of Experimental Biology 208 (14): p2673-2682 JUL 2005 2005
ISSN: 0022-0949
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have cloned a homologue of mammalian aquaporin-3 (AQP3) from gills of Mozambique tilapia using a reverse transcription-polymerase chain reaction (RT-PCR). The deduced amino acid sequence shared 64-75% homology with other vertebrate AQP3 homologues. RT-PCR revealed that tilapia AQP3 was expressed in the brain, pituitary, kidney, spleen, intestine, skin, eye and gill in tilapia adapted to freshwater (FW) and seawater (SW). We also examined functional characteristics of tilapia AQP3 using Xenopus oocytes as an in vitro transcribed cRNA expression system. Osmotic water permeability (Pf) of Xenopus oocytes expressing tilapia AQP3 was about 30-fold higher than that of control oocytes, and was 80% inhibited by treatment with 0.3 mmol l(-1) HgCl2. Light-microscopic %%%immunocytochemistry%%% of branchial epithelia revealed that tilapia AQP3 was expressed in gill chloride cells of FW- and SW-adapted tilapia. Electron-microscopic %%%immunocytochemistry%%% further demonstrated that tilapia AQP3 was localized in the basolateral membrane of gill chloride cells. Basolateral localization of AQP3 in gill chloride cells suggests that AQP3 is involved in regulatory volume changes and osmoreception, which could trigger functional differentiation of chloride cells.

2/7/14 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18468533 BIOSIS NO.: 200510163033
Expression and %%%immunolocalization%%% of water-channel aquaporins in the rat and mouse mammary gland
AUTHOR: Matsuzaki Toshiyuki (Reprint); Machida Natsuko; Tajika Yuki;

Ablimit Abdushukur; Suzuki Takeshi; Aoki Takeo; Hagiwara Haruo; Takata Kuniaki
AUTHOR ADDRESS: Gunma Univ, Grad Sch Med, Dept Anat and Cell Biol, Showa Machi 3-39-22, Gunma 3718511, Japan**Japan
AUTHOR E-MAIL ADDRESS: matoshi@med.gunma-u.ac.jp
JOURNAL: Histochemistry and Cell Biology 123 (4-5): p501-512 JUN 2005 2005
ISSN: 0948-6143
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We examined the expression and %%%immunolocalization%%% of water-channel aquaporins in the mammary gland by reverse transcriptase polymerase chain reaction (RT-PCR), %%%immunoblotting%%%, and %%%immunohistochemistry%%%. RT-PCR and %%%immunoblotting%%% revealed the expression of aquaporin-1 (AQP1) and AQP3 in the lactating rat mammary gland. AQP3 was detected in the alveolar epithelium and duct system whereas AQP1 was found in the capillaries and venules. AQP3 was present in the basolateral membrane of secretory epithelial cells and intralobular and interlobular duct epithelial cells. The main duct near the orifice in the nipple, which is comprised of a stratified epithelium, bore AQP3 in its basal and intermediate layers. AQP1 was located in both the apical and basolateral membranes of capillary and venule endothelia. AQP3 was not detected in virgin females. AQP3 was found in some differentiating mammary epithelial cells in the pregnant rat. AQP1 was present in capillaries and venules in the differentiating mammary gland of the pregnant rat and in the mammary fat pad of virgin females. We found a similar distribution of AQP1 and AQP3 in the mouse. AQP1 and AQP3 seem to play roles in the synthesis and/or secretion of milk.

2/7/15 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17741716 BIOSIS NO.: 200400111422
Increased renal ENaC subunit and sodium transporter abundances in streptozotocin-induced type 1 diabetes.
AUTHOR: Song Jian; Knepper Mark A; Verbalis Joseph G; Ecelbarger Carolyn A (Reprint)
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JOURNAL: American Journal of Physiology 285 (6 Part 2): pF1125-F1137 December 2003 2003
MEDIUM: print
ISSN: 0002-9513 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Uncontrolled diabetes mellitus (DM) is associated with copious water and sodium losses. We hypothesized that the kidney compensates for these losses by increasing the abundances of key sodium and water transporters and channels. Using targeted proteomic analysis via %%%immunoblotting%%% of kidney homogenates, we examined comprehensive regulation of transport proteins. In three studies, streptozotocin (STZ;

65 mg/kg) or vehicle was administered intraperitoneally to male Sprague-Dawley rats. In study 2, to control for potential renal toxicity of STZ, one group of STZ-treated rats was intensively treated with insulin to control diabetes. In another group, the reversibility of DM and related changes was assessed by treating animals with insulin for the final 4 days. In study 3, we correlated blood glucose to transporter changes by treating animals with different doses of insulin. In study 1, STZ treatment resulted in significantly increased band densities for the type 3 sodium/hydrogen exchanger (NHE3), the thiazide-sensitive Na-Cl cotransporter (NCC), and epithelial sodium channel (ENaC) subunits alpha, beta, and gamma (85- and 70-kDa bands) to 204, 125, 176, 132, 147, and 241% of vehicle mean, respectively. In study 2, aquaporin-2 (AQP2) and AQP3 were increased with DM, but not AQP1 or AQP4. Neither these changes, nor blood glucose itself, could be returned to normal by short-term intensive insulin treatment. Whole kidney abundance of AQP3, the bumetanide-sensitive Na-K-2Cl cotransporter (NKCC2), and gamma-ENaC (85-kDa band) correlated most strongly with blood glucose in study 3. These comprehensive changes would be expected to decrease volume contraction accompanying large-solute and water losses associated with DM.

2/7/16 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17656443 BIOSIS NO.: 200400027200
Estrogen regulation of aquaporins in the mouse uterus: Potential roles in uterine water movement.
AUTHOR: Jablonski Elizabeth M; McConnell Nisha A; Hughes Francis M; Huet-Hudson Yvette M (Reprint)
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JOURNAL: Biology of Reproduction 69 (5): p1481-1487 November 2003 2003
MEDIUM: print
ISSN: 0006-3363
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Estrogen stimulates water imbibition in the uterine endometrium. This water then crosses the epithelial cells into the lumen, leading to a decrease in viscosity of uterine luminal fluid. To gain insight into the mechanisms underlying this estrogen-stimulated water transport, we have explored the expression profile and functionality of water channels termed aquaporins (AQPs) in the ovariectomized mouse uterus treated with ovarian steroid hormones. Using %%%immunocytochemical%%% analysis and %%%immunoprecipitation%%% techniques, we have found that AQP-1, -3, and -8 were constitutively expressed. AQP-1 expression was restricted to the myometrium and may be slightly regulated by ovarian steroid hormones. AQP-3 was expressed at low levels in the epithelial cells and myometrium, whereas AQP-8 was found in both the stromal cells and myometrium. AQP-2 was absent in vehicle controls but strongly up-regulated by estrogen in the epithelial cells and myometrium of the uterus. This localization implicates all four isotypes in movement of water during uterine imbibition and, based on their localization to the luminal epithelial

cells, AQP-2 and -3 in facilitating water movement into the lumen of the uterus. The analysis of the plasma membrane permeability of luminal epithelial cells by two separate cell swelling assays confirmed a highly increased water permeability of these cells in response to estrogen treatment. This finding suggests that estrogen decreases the luminal fluid viscosity, in part, by enhancing the water permeability of the epithelial layer, most likely by increasing the expression of AQP-2 and/or the availability of AQP-3. Together these results provide novel information concerning the mechanism by which estrogen controls water imbibition and luminal fluid viscosity in the mouse uterus.

2/7/17 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17528190 BIOSIS NO.: 200300485847
Aquaporin expression in normal human kidney and in renal disease.
AUTHOR: Bedford Jennifer J; Leader John P; Walker Robert J (Reprint)
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JOURNAL: Journal of the American Society of Nephrology 14 (10): p2581-2587 October 2003 2003
MEDIUM: print
ISSN: 1046-6673
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aquaporins (AQPs), membrane-inserted water channel proteins, play a highly important role in the reabsorption of water from the renal tubular fluid. Experimentally, both in rats and mice, failure to insert functional AQP molecules into renal tubular membranes leads to nephrogenic diabetes insipidus. In humans, most forms of renal disease lead to a reduction in the water handling capacity of the kidney. AQP distribution in various forms of human renal disease has not been documented. %%%Immunohistochemical%%% studies of biopsy samples from a wide range of renal diseases revealed a substantial and striking upregulation of AQP-1 in the glomeruli of most diseased kidneys. AQP-1 expression remained prominent in proximal tubules in all lesions. In contrast, there was judged qualitatively to be a reduction in the amounts of AQP-2 and AQP-3 expression, especially in lesions with substantial interstitial fibrosis and nephron loss, as compared with a healthy region of normal kidneys. The results were quantitatively confirmed by real-time reverse transcriptase-PCR. This is the first documentation of altered AQP expression in human renal disease. The significance of the increased AQP-1 expression requires further studies.

2/7/18 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17471955 BIOSIS NO.: 200300426799
Analysis of the expression of aquaporin-1 and aquaporin-9 in pig liver tissue: Comparison with rat liver tissue.

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JOURNAL: Cells Tissues Organs 174 (3): p117-128 2003 2003
MEDIUM: print
ISSN: 1422-6405
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aquaporins (AQPs) are cellular proteins involved with the movement of water across cell membranes and are fundamentally important to the fluid transport in the bile ducts and ductules of the liver. An %%%immunohistochemical%%% analysis of AQP-1 and AQP-9 was undertaken to describe their expression in fetal and adult pig liver, while %%%immunoreagents%%% specific to some other AQPs were screened for their efficacy on pig liver tissues. Anti-AQP-1 antibody reacted with the bile duct of the portal space and the bile ductules at the periphery of the liver lobules. Histological identification of bile ductules was confirmed by positive reactivity with anti-cytokeratin-7 and antilaminin %%%immunostaining%%%. Anti-AQP-1 signals were also pronounced in the endothelium of the portal space blood vessels and peripheral distributing venules. Antibody to AQP-9 reacted strongly with small ductules peripheral to the liver lobules, but only weakly with the bile ducts of the portal space. Anti-AQP-adipose antibody bound to the smooth muscle cells of the arteries in the portal space and sporadically with certain binucleated cells in the liver lobule. Antibodies to AQP-3, AQP-4, AQP-7, and AQP-8 were nonreactive with any of the tissues of the adult pig liver. For comparative purposes, %%%immunohistochemical%%% analysis of rat liver tissue was done with the anti-AQP-1 and AQP-9 antibodies. Anti-AQP-1 reacted weakly with the rat liver's bile ducts, but robustly with the endothelium of the liver's veins and arteries. It also reacted strongly with the central vein of the rat liver lobules, and, because the staining was continuous with hepatic sinusoids, it appeared that the reactivity was specific to the endothelial cells. Anti-AQP-9 antibodies reacted with rat hepatocytes and was not associated with the canaliculi, as judged by concurrent phalloidin staining of actin. The results indicate that specific AQPs are expressed in the tissues of the pig liver and that AQP-9 expression is distinct from its expression in the rat liver.

2/7/19 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17406891 BIOSIS NO.: 200300365610
Estrogen regulation of the expression and function of AQPs in the mouse uterus.
AUTHOR: Jablonski Elizabeth M (Reprint); McConnell Nisha A (Reprint); Motameni Amirreza T (Reprint); Huet-Hudson Yvette M (Reprint); Hughes Francis M (Reprint)
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JOURNAL: Biology of Reproduction 68 (Supplement 1): p117-118 2003 2003

MEDIUM: print
CONFERENCE/MEETING: Thirty-sixth Annual Meeting of the Society for the Study of Reproduction Cincinnati, OH, USA July 19-22, 2003; 20030719
SPONSOR: Society for the Study of Reproduction
ISSN: 0006-3363
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/20 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17371877 BIOSIS NO.: 200300330173
Downregulation of renal aquaporins in response to unilateral ureteral obstruction.
AUTHOR: Li Chunling; Wang Weidong; Knepper Mark A; Nielsen Soren; Frokiaer Jorgen (Reprint)
AUTHOR ADDRESS: The Water and Salt Research Ctr., Institute of Experimental Clinical Research, Aarhus Univ. Hospital-Skejby, DK-8200, Aarhus, Denmark **Denmark
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JOURNAL: American Journal of Physiology 284 (5 Part 2): pF1066-F1079 May 2003 2003
MEDIUM: print
ISSN: 0002-9513 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The expression of aquaporin-2 (AQP2) is decreased in rats with bilateral ureteral obstruction (BUO) and unilateral ureteral obstruction (UUO). Therefore, the expression of additional renal aquaporins (AQP1-4) and phosphorylated AQP2 (p-AQP2), known to play a role in urinary concentration, was examined in a Wistar rat model with 24 h of UUO. In obstructed kidneys, %%%immunoblotting%%% revealed a significant decrease in the expression of inner medullary AQP2 to 42 +- 4, p-AQP2 to 23 +- 5, AQP3 to 19 +- 6, AQP4 to 11 +- 5, and AQP1 to 64 +- 8% of sham levels. AQP1 expression located in the proximal tubule decreased to 74 +- 4% of sham levels ($P < 0.05$). %%%Immunocytochemistry%%% confirmed the downregulation of AQP3, AQP4, and p-AQP2. In contralateral nonobstructed kidneys, %%%immunoblotting%%% also revealed significant reductions of AQP1 in the inner medulla, outer medulla, and cortex, whereas expression of AQP2, AQP3, AQP4, and p-AQP2 was unchanged. Furthermore, we collected the urine from both obstructed and nonobstructed kidneys for 2 h, respectively, after 24 h of UUO. Urine collection from obstructed kidneys during 2 h after release of UUO revealed a significant reduction in urine osmolality and solute-free water reabsorption (TcH2O). Moreover, an increase in urine production and TcH2O was observed in contralateral kidneys. To examine whether vasopressin-independent mechanisms are involved in AQP2 regulation, vasopressin-deficient Brattleboro (BB) rats with 24 h of UUO were examined. %%%Immunoblotting%%% revealed downregulation of AQP2, p-AQP2, AQP3, and AQP1 in obstructed kidneys and downregulation of p-AQP2 and AQP1 in nonobstructed kidneys. In conclusion, 1) UUO is associated with severe downregulation of AQP2, AQP3, AQP4, and AQP1; thus all of these AQPs may play important roles in

the impaired urinary concentrating capacity in the obstructed kidney; 2) the reduced levels of AQP1 in the nonobstructed kidney may contribute to the compensatory increase in urine production; and 3) downregulation of AQPs in BB rats supports the view that vasopressin-independent pathways may be involved in AQP2 and AQP3 regulation in the obstructed kidney.

2/7/21 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17371838 BIOSIS NO.: 200300330134
Mechanism of acid adaptation of a fish living in a pH 3.5 lake.
AUTHOR: Hirata Taku; Kaneko Toyoji; Ono Toshihiro; Nakazato Takeru; Furukawa Norihisa; Hasegawa Sanae; Wakabayashi Shigeo; Shigekawa Munekazu ; Chang Min-Hwang; Romero Michael F; Hirose Shigehisa (Reprint)
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JOURNAL: American Journal of Physiology 284 (5 Part 2): pR1199-R1212 May 2003 2003
MEDIUM: print
ISSN: 0002-9513 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Despite unfavorable conditions, a single species of fish, Osorezan dace, lives in an extremely acidic lake (pH 3.5) in Osorezan, Aomori, Japan. Physiological studies have established that this fish is able to prevent acidification of its plasma and loss of Na+. Here we show that these abilities are mainly attributable to the chloride cells of the gill, which are arranged in a follicular structure and contain high concentrations of Na+-K+-ATPase, carbonic anhydrase II, type 3 Na+/H+ exchanger (NHE3), type 1 Na+-HCO3- cotransporter, and aquaporin-3, all of which are upregulated on acidification. %%%Immunohistochemistry%%% established their chloride cell localization, with NHE3 at the apical surface and the others localized to the basolateral membrane. These results suggest a mechanism by which Osorezan dace adapts to its acidic environment. Most likely, NHE3 on the apical side excretes H+ in exchange for Na+, whereas the electrogenic type 1 Na+-HCO3- cotransporter in the basolateral membrane provides HCO3- for neutralization of plasma using the driving force generated by Na+-K+-ATPase and carbonic anhydrase II. Increased expression of glutamate dehydrogenase was also observed in various tissues of acid-adapted dace, suggesting a significant role of ammonia and bicarbonate generated by glutamine catabolism.

2/7/22 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17302359 BIOSIS NO.: 200300261003
AQP3-expressing %%%immune%%% cells in erythema toxicum neonatorum: A possible role in %%%immunosurveillance%%% at birth.
AUTHOR: Marchini Giovanna (Reprint); Kankes Kanchana; Stabi Berit;

Lonne-Rahm Solbritt; Nielsen Soren
AUTHOR ADDRESS: Woman and Child Health, Astrid Lindgren Children's Hospital, Karolinska Institutet, Stockholm, Sweden**Sweden
JOURNAL: Pediatric Research 53 (4 Part 2): p363A April 2003 2003
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Pediatric Academic Societies' Seattle, WA, USA May 03-06, 2003; 20030503
SPONSOR: Pediatric Academic Societies
ISSN: 0031-3998 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Paper; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

2/7/23 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17156201 BIOSIS NO.: 200300114920
cDNA array identification of genes regulated in rat renal medulla in response to vasopressin infusion.
AUTHOR: Brooks Heddwen L; Ageloff Shana; Kwon Tae-Hwan; Brandt William; Terris James M; Seth Akhil; Michea Luis; Nielsen Soren; Fenton Robert; Knepper Mark A (Reprint)
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JOURNAL: American Journal of Physiology 284 (1 Part 2): pF218-F228 January 2003 2003
MEDIUM: print
ISSN: 0002-9513 _(ISSN print)
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: With the aim of identifying possible gene targets for direct or indirect regulation by vasopressin in the renal medulla, we have carried out cDNA array experiments in inner medullas of Brattleboro rats infused with the V2 receptor-selective vasopressin analog desamino-Cys1,D-Arg8 vasopressin (dDAVP) for 72 h. Of the 1,176 genes on the array, 137 transcripts were increased by 2-fold or more, and 10 transcripts were decreased to 0.5-fold or less. Quantitative, real-time RT-PCR measurements confirmed increases seen for six selected transcripts (Wilms' tumor protein, beta-arrestin 2, neurofibromin, casein kinase IIbeta, aquaporin-3, and aquaporin-4). To correlate changes in mRNA expression with changes in protein expression, we carried out quantitative %%%immunoblotting%%% for 28 of the proteins whose cDNAs were on the array. For several targets including aquaporin-2, transcript abundance and protein abundance changes did not correlate. However, for most genes examined, changes in mRNA abundances were associated with concomitant protein abundance changes. Targets with demonstrated increases in both protein and mRNA abundances included neurofibromin, casein kinase IIbeta, the beta-subunit of the epithelial Na channel (beta-ENaC), 11beta-hydroxysteroid dehydrogenase type 2, and c-Fos. Additional cDNA arrays revealed that several transcripts that were increased in abundance after 72 h of dDAVP were also increased after 4 h, including casein kinase IIbeta, beta-ENaC, aquaporin-3, UT-A, and

syntaxin 2. These studies have identified several transcripts whose abundances are regulated in the inner medulla in response to infusion of dDAVP and that could play roles in the regulation of salt and water excretion.

2/7/24 (Item 23 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17111625 BIOSIS NO.: 200300070344
Regulation of collecting duct AQP3 expression: Response to mineralocorticoid.
AUTHOR: Kwon Tae-Hwan; Nielsen Jakob; Masilamani Shyama; Hager Henrik; Knepper Mark A; Frokiaer Jorgen; Nielsen Soren (Reprint)
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JOURNAL: American Journal of Physiology 283 (6 Part 2): pF1403-F1421 December 2002 2002
MEDIUM: print
ISSN: 0002-9513 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adrenocortical steroid hormones are importantly involved in the regulation of extracellular fluid volume. The present study was aimed at examining whether aldosterone and/or glucocorticoid regulates the abundance of aquaporin-3 (AQP3), -2, and -1 in rat kidney. In protocol 1, rats were adrenalectomized, followed by aldosterone replacement, dexamethasone replacement, or combined aldosterone and dexamethasone replacement (rats had free access to water but received a fixed amount of food). Protocol 2 was identical to protocol 1, except that all groups received fixed daily food and water intake. In both protocols 1 and 2, aldosterone deficiency was associated with increased fractional Na excretion and severe hyperkalemia. Semiquantitative %%%immunoblotting%%% revealed that aldosterone deficiency was associated with a dramatic downregulation of AQP3 abundance. Consistent with this, %%%immunocytochemistry%%% and %%%immunoelectron%%% microscopy revealed a marked decrease in AQP3 labeling in the basolateral plasma membranes of collecting duct principal cells. In contrast, AQP1 and AQP2 abundance and distribution were unchanged. Glucocorticoid deficiency revealed no changes in AQP3, -2, or -1 abundance. In protocol 3, Na restriction (to increase endogenous aldosterone levels) or exogenous aldosterone infusion in either normal rats or vasopressin-deficient Brattleboro rats was associated with a major increase in AQP3 abundance. In protocol 4, aldosterone levels were clamped by infusion of aldosterone, while Na intake was altered from a low to a high level. Under these circumstances, there were no changes in AQP3 or AQP2 abundance, although the level of the thiazide-sensitive Na-Cl cotransporter was decreased. In conclusion, the results uniformly demonstrate that aldosterone regulates AQP3 abundance independently of Na intake. In contrast, changes in glucocorticoid levels in these models do not influence AQP3 or AQP2 abundance. Therefore, in the collecting duct aldosterone may regulate, at least in part, AQP3 expression in addition to regulating Na and K transport.

2/7/25 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17029051 BIOSIS NO.: 200200622562
Selective down-regulation of aquaporin-1 in salivary glands in primary Sjogren's syndrome
AUTHOR: Beroukas Dimitra; Hiscock Jenny; Gannon Bren J; Jonsson Roland; Gordon Tom P; Waterman Sally A (Reprint)
AUTHOR ADDRESS: Department Immunology, Allergy and Arthritis, School of Medicine, Flinders University, GPO Box 2100, Adelaide, SA, 5001, Australia**Australia
JOURNAL: Laboratory Investigation 82 (11): p1547-1552 November, 2002 2002
MEDIUM: print
ISSN: 0023-6837
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Salivary and lacrimal gland secretions are reduced in primary Sjogren's syndrome (pSS). Aquaporins (AQPs) are involved in transmembrane water transport, and different isoforms show specific cellular and subcellular distributions in salivary and lacrimal glands. Changes in expression of AQP molecules have therefore been suggested to contribute to the glandular dysfunction in pSS. AQP-5 is present in the apical membrane of acinar cells, where it mediates fluid outflow; however, we have recently shown that its expression is not altered in pSS. We therefore studied whether expression of other isoforms of AQP would be altered in pSS. Using high-resolution confocal microscopy, we determined the distribution of AQP-1 and AQP-3 in labial salivary gland biopsies from 11 patients with pSS and 9 healthy controls. AQP-1 is present in myoepithelial cells surrounding acini, and its expression in these cells was decreased by 38% in pSS glands. By contrast, expression of AQP-1 in endothelial cells of nonfenestrated capillaries was not altered in pSS. AQP-3 was expressed in the basolateral membrane of acinar epithelial cells, and its expression was not altered in disease. We therefore conclude that AQP-1 expression in myoepithelial cells is selectively down-regulated in pSS and that myoepithelial cell dysfunction may play a crucial role in the pathology of this disease.

2/7/26 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16958288 BIOSIS NO.: 200200551799
Expression of aquaporin-3 in human peritoneal mesothelial cells and its up-regulation by glucose in vitro
AUTHOR: Lai Kar Neng (Reprint); Leung Joseph C K; Chan Loretta Y Y; Tang Sydney; Li Fu Keung; Lui Sing Leung; Chan Tak Mao
AUTHOR ADDRESS: Department of Medicine, University of Hong Kong, Queen Mary Hospital, 102 Pokfulam Road, Room 409, Professorial Block, Hong Kong, China**China
JOURNAL: Kidney International 62 (4): p1431-1439 October, 2002 2002
MEDIUM: print

ISSN: 0085-2538
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Aquaporin-3 (AQP3) is a member of the water channel family that is selective for the passage of not only water, but also glycerol and urea. Our recent study demonstrated the presence of aquaporin-1 in human peritoneal mesothelial cells (HPMC). Although transcripts encoding for AQP3 has been detected by reverse transcription-polymerase chain reaction (RT-PCR) in murine peritoneal mesothelium, to date there is no documentation of protein expression on peritoneal mesothelial cells. Method: Our present study was designed to explore the gene and protein expression of AQP3 in HPMC and its regulation under different concentrations of glucose. Results: AQP3 protein was detected in the human peritoneal tissue by %%%immunohistological%%% staining using specific, affinity-purified polyclonal anti-AQP3 antibodies. AQ3 transcripts and protein expression in cultured HPMC were investigated by RT-PCR and %%%immunoblotting%%% analysis respectively. Cell permeability to glycerol (flux) was measured using (14C)glycerol incorporation. AQP3 transcript and protein were weakly expressed in HPMC constitutively. The gene expression of AQP3 and its protein biosynthesis in HPMC were inducible following exposure to glucose in a dose- and time-dependent manner (P<0.0001). Glucose at a concentration of 200 mmol induced glycerol flux by 4.82-fold above the control value (P<0.0001) and its effect was significantly inhibited by mercuric chloride (P<0.01). Conclusion: Our novel observation demonstrated the AQP3 expression and biosynthesis in HPMC and in vitro studies revealed that glycerol permeability in HPMC was up-regulated by glucose. Further study is warranted to elucidate the role of AQP3 in HPMC for maintaining the ultrafiltration of the peritoneal membrane.

16935059 BIOSIS NO.: 200200528570
Distribution of aquaporins in the colon of Octodon degus, a South American desert rodent
AUTHOR: Gallardo Pedro (Reprint); Olea Nancy; Sepulveda Francisco V
AUTHOR ADDRESS: ICBM Facultad de Medicina, Universidad de Chile, Casilla 70058, Santiago, Chile**Chile
JOURNAL: American Journal of Physiology 283 (3 Part 2): pR779-R788
September, 2002 2002
MEDIUM: print
ISSN: 0002-9513
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Octodon degus is a desert rodent of northern Chile, adapted to survive with a limited supply of water. This rodent has a high degree of fecal dehydration, related to colon water absorption. With the hypothesis that aquaporins (AQPs) might be present in the colon epithelium of O. degus and involved in fluid absorption, we studied colon water absorption in vivo and the distribution of AQPs and Na+ transporters by

%%%immunocytochemistry%%%. AQP-1 was found in apical and basolateral membranes of surface-absorptive and crypt epithelial cells. AQP-8 was found in the cytoplasm of enterocytes of surface colon. AQP-3 %%%immunolabeling%%%, on the other hand, was absent from the epithelium but present in a subepithelial fibroblast layer, pericryptal cells, and muscularis mucosae. The hydration state did not modify the amount of %%%immunostaining%%% for any of the AQPs. Colon water absorption was markedly decreased by the mercurial agent p-chloromeri-curibenzenesulfonic acid and was not affected by water deprivation. The NHE3 isoform of Na+/H+ exchanger and alpha-1 subunit of the Na+-K+-ATPase were found in apical and basolateral membranes of surface-cabsorptive cells, respectively. These results suggest that colon water absorption is mostly transcellular and mediated by water channels like AQP-1. Apical Na+/H+ exchanger and basolateral Na+-K+-ATPase in surface cells could be part of the Na+ absorption pathway. It is hypothesized that this transport is necessary to provide an osmotic gradient for water absorption. The roles of AQP-8 and AQP-3 in water absorption remain to be established.

16790429 BIOSIS NO.: 200200383940
Expression, localization, and regulation of aquaporin-1 to -3 in rat urothelia
AUTHOR: Spector David A (Reprint); Wade James B; Dillow Russell; Steplock Deborah A; Weinman Edward J
AUTHOR ADDRESS: Div. of Renal Medicine, Johns Hopkins Bayview Medical Ctr., 4940 Eastern Ave., B2N, Baltimore, MD, 21224, USA**USA
JOURNAL: American Journal of Physiology 282 (6 Part 2): pF1034-F1042 June, 2002 2002
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ABSTRACT: Although mammalian urothelia are generally considered impermeable to constituents of urine, in vivo studies in several species indicate urothelial transport of water and solutes under certain conditions. This study investigates the expression, localization, and regulation of aquaporin (AQP)-1, -2, and -3 in ureteral and bladder tissues in 48-h dehydrated and water-loaded female Wistar rats. %%%Immunoblots%%% of homogenates of whole ureter and bladder identified characteristics apprx28- and 35- to 44-kDa bands for AQP-1, -2, and -3. AQP-1 was localized to capillary and arteriole endothelial cells, whereas AQP-2 and -3 circumferentially lined the epithelial cell membranes except for the apical membrane of the epithelial cells adjacent to the lumens of both ureter and bladder. AQP-2 was also present in epithelial cell cytoplasm. Dehydration resulted in 160-200% increases of AQP-3 signal and 24-49% increases of AQP-2 signal but no change in AQP-1 signal on %%%immunoblots%%% of homogenates of ureters and bladders. AQPs in genitourinary tract urothelia likely play a role in the regulation of epithelial cell volume and osmolality and may play a role in bulk water movement across urothelia.

2/7/29 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16753027 BIOSIS NO.: 200200346538
Impaired stratum corneum hydration in mice lacking epidermal water channel aquaporin-3
AUTHOR: Ma Tonghui (Reprint); Hara Mariko; Sougrat Rachid; Verbavatz Jean-Marc; Verkman A S
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JOURNAL: Journal of Biological Chemistry 277 (19): p17147-17153 May 10, 2002 2002
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The water and solute transporting properties of the epidermis have been proposed to be important determinants of skin moisture content and barrier properties. The water/small solute-transporting protein aquaporin-3 (AQP3) was found by %%%immunofluorescence%%% and %%%immunogold%%% electron microscopy to be expressed at the plasma membrane of epidermal keratinocytes in mouse skin. We studied the role of AQP3 in stratum corneum (SC) hydration by comparative measurements in wild-type and AQP3 null mice generated in a hairless SKH1 genetic background. The hairless AQP3 null mice had normal perinatal survival, growth, and serum chemistries but were polyuric because of defective urinary concentrating ability. AQP3 deletion resulted in a >4-fold reduced osmotic water permeability and >2-fold reduced glycerol permeability in epidermis. Epidermal, dermal, and SC thickness and morphology were not grossly affected by AQP3 deletion. Surface conductance measurements showed remarkably reduced SC water content in AQP3 null mice in the hairless genetic background (165+-10 versus 269+-12 microsiemens (muS), p<0.001), as well as in a CD1 genetic background (209+-21 versus 469+-11 muS). Reduced SC hydration was seen from 3 days after birth. SC hydration in hairless wild-type and AQP3 null mice was reduced to comparable levels (90-100 muS) after a 24-h exposure to a dry atmosphere, but the difference was increased when surface evaporation was prevented by occlusion or exposure to a humidified atmosphere (179+-13 versus 441+-34 muS). Conductance measurements after serial tape stripping suggested reduced water content throughout the SC in AQP3 null mice. Water sorption-desorption experiments indicated reduced water holding capacity in the SC of AQP3 null mice. The impaired skin hydration in AQP3 null mice provides the first functional evidence for the involvement of AQP3 in skin physiology. Modulation of AQP3 expression or function may thus alter epidermal moisture content and water loss in skin diseases.

2/7/30 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16589653 BIOSIS NO.: 200200183164
Differential gene expression profiling in human brain tumors
AUTHOR: Markert James M; Fuller Catherine M; Gillespie G Yancey; Bubien James K; McLean Lee Anne; Hong Robert L; Lee Kailin; Gullans Steven R; Mapstone Timothy B; Benos Dale J (Reprint)
AUTHOR ADDRESS: Dept. of Physiology and Biophysics, Univ. of Alabama at Birmingham, 1918 Univ. Boulevard, MCLM 704, Birmingham, AL, 35294-0005, USA**USA
JOURNAL: Physiological Genomics 5 p21-33 June, 2001 2001
MEDIUM: print
ISSN: 1094-8341
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Gene expression profiling of three human temporal lobe brain tissue samples (normal) and four primary glioblastoma multiforme (GBM) tumors using oligonucleotide microarrays was done. Moreover, confirmation of altered expression was performed by whole cell patch clamp, %%%immunohistochemical%%% staining, and RT-PCR. Our results identified several ion and solute transport-related genes, such as N-methyl-D-aspartate (NMDA) receptors, alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionate (AMPA)-2 receptors, GABAA receptor subunits alpha3, beta1, beta2, and beta3, the glutamate transporter, the glutamate/aspartate transporter II, the potassium channel KV2.1, hKVbeta3, and the sodium/proton exchanger 1 (NHE-1), that are all downregulated in the tumors compared with the normal tissues. In contrast, aquaporin-1, possibly aquaporins-3 and -5, and GLUT-3 message appeared upregulated in the tumors. Our results also confirmed previous work showing that osteopontin, nicotinamide N-methyltransferase, murine double minute 2 (MDM2), and epithelin (granulin) are upregulated in GBMs. We also demonstrate for the first time that the cytokine and p53 binding protein, macrophage migration inhibitory factor (MIF), appears upregulated in GBMs. These results indicate that the modulation of ion and solute transport genes and heretofore unsuspected cytokines (i.e., MIF) may have profound implications for brain tumor cell biology and thus may identify potential useful therapeutic targets in GBMs.

 2/7/31 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16186044 BIOSIS NO.: 200100357883
Downregulation of AQP1, -2, and -3 after ureteral obstruction is associated with a long-term urine-concentrating defect
AUTHOR: Li Chunling; Wang Weidong; Kwon Tae-Hwan; Isikay Levent; Wen Jian Guo; Marples David; Djurhuus Jens Christian; Stockwell Anette; Knepper Mark A; Nielsen Soren; Frokiaer Jorgen (Reprint)
AUTHOR ADDRESS: Institute of Experimental Clinical Research, Aarhus Univ. Hospital-Skejby, DK-8200, Aarhus, Denmark**Denmark
JOURNAL: American Journal of Physiology 281 (1 Part 2): pF163-F171 July, 2001 2001
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Previously, we demonstrated that 24 h of bilateral ureteral obstruction (BUO) and short-term release of BUO was associated with a decrease in the expression of aquaporin-2 (AQP2), polyuria, and a reduced urinary concentrating capacity (10). The purposes of the present study were to examine whether BUO and the long-term release of BUO (BUO-R) for 3, 14, and 30 days were associated with changes in the expression of renal AQP1, AQP2, and AQP3 and whether such changes were associated with parallel changes in urinary output and urinary concentrating capacity. Rats (n=4-7 in each group) were kept in metabolic cages for measurements of urinary output. Kidneys were removed to determine the expression levels of AQP1, AQP2, and AQP3 by semiquantitative %%%immunoblotting%%%. AQP2 was downregulated after 24 h of BUO (42+-3%). Downregulation of AQP2 persisted 3 (43+-14%; P<0.01) and 15 days after BUO-R (48+-11%; P<0.01) but was normalized 30 days after BUO-R. AQP3 showed a similar pattern. Moreover, AQP1 was downregulated in response to BUO (65+-7%) and remained downregulated 3 days after BUO-R (41+-5%), 14 days after BUO-R (57+-8%), and 30 days after BUO-R (59+-5%). BUO-R resulted in a significant polyuria that gradually decreased, although it remained significant at day 30. Urinary concentrating capacity remained significantly impaired when determined 3, 14, and 30 days after BUO-R in response to a 24-h period of thirst (1,712+-270 vs. 2,880+-91 mosmol/kgH2O at day 30, P<0.05). In conclusion, the expression of AQP1, AQP2, and AQP3 were long-term downregulated after BUO-R, suggesting that dys-regulation of aquaporins located at the proximal tubule, thin descending limb of the loop of Henle, and the collecting duct may contribute to the long-term polyuria and impairment of urinary concentrating capacity associated with obstructive nephropathy.

2/7/32 (Item 31 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16136065 BIOSIS NO.: 200100307904
Alterations in the expression of the AQP family in cultured rat astrocytes during hypoxia and reoxygenation
AUTHOR: Yamamoto Naoki; Yoneda Kazuhiro; Asai Kiyofumi (Reprint); Sobue Kazuya; Tada Toyohiro; Fujita Yoshihito; Katsuya Hirotada; Fujita Masataka; Aihara Noritaka; Mase Mitsuhito; Yamada Kazuo; Miura Yutaka; Kato Taiji
AUTHOR ADDRESS: Department of Bioregulation Research, Nagoya City University Medical School, Mizuho-ku, Nagoya, 467-8601, Japan**Japan
JOURNAL: Molecular Brain Research 90 (1): p26-38 20 May, 2001 2001
MEDIUM: print
ISSN: 0169-328X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aquaporins (AQPs) are a family of water-selective transporting proteins with homology to the major intrinsic protein (MIP) of lens (Cells 39 (1984) 49), that increase plasma membrane water permeability in secretory and absorptive cells. In the central nervous system (CNS), we detected the transcripts of AQP3, 5 and 8 in addition to the previously reported transcripts of AQP4 and 9 in astrocytes, of AQP3, 5 and 8 in

neurons, of AQP8 in oligodendrocytes, and none of them in microglia using RNase protection assay and the reverse transcription-polymerase chain reaction (RT-PCR). Hypoxia evoked a marked decrease in the expression levels of AQP4, 5 and 9, but not of AQP3 and 8 mRNAs, and in astrocytes in vitro subsequent reoxygenation elicited the restoration of the expression of AQP4 and 9 to their basal levels. Interestingly, AQP5 showed a transient up-regulation (about 3-fold) and subsequent down-regulation of its expression within 20 h reoxygenation after hypoxia. The changes in the profiles of AQP expression during hypoxia and reoxygenation were also observed by Western blot analysis. These results suggest that AQP5 may be one of the candidates for inducing the intracranial edema in the CNS after ischemia injury.

16086256 BIOSIS NO.: 200100258095
Spironolactone-mediated downregulation of the epithelial sodium channel (ENaC) in rat kidney
AUTHOR: Nielsen Jakob (Reprint); Beutler Kathleen T (Reprint); Knepper Mark A (Reprint)
AUTHOR ADDRESS: NIH, 10 Center Drive, MSC 1603, Bethesda, MD, 20892-1603, USA**USA
JOURNAL: FASEB Journal 15 (4): pA432 March 7, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The epithelial Na channel (ENaC) of the renal collecting duct is an important molecular target for regulation of renal tubular Na transport by aldosterone. Aldosterone administration or dietary NaCl restriction in rats markedly increased the abundance of the alpha subunit (but not the beta and gamma subunits) of ENaC (Masilamani et al. J.Clin.Invest. 104:R19-23, 1999). In addition, aldosterone caused a shift in the molecular weight of gamma ENaC from 85 to 70 kDa, putatively due to a physiological proteolytic cleavage of the protein. Aldosterone is known to have both genomic and nongenomic effects on cells. The genomic effects are mediated by the mineralocorticoid receptor (MR). To test whether the demonstrated changes in ENaC in response to dietary salt restriction are mediated by the MR, we carried out semiquantitative %%%immunoblotting%%% of whole kidney homogenates from NaCl-restricted rats. Spironolactone was given in the food at 350 mg/kgBW/day for 7 days. The intake of calories, protein, NaCl and water were equalized in spironolactone-treated and control rats by ration feeding. Spironolactone-treated rats exhibited a marked decrease in alpha ENaC abundance (57+-9% of control, P<0.05) with no significant change in the total abundances beta and gamma ENaC. However, there was a significant decrease in the abundance of the 70 kDa form of gamma ENaC (60+-6% of control, P<0.05) with a significant increase in the abundance of the 85 kDa form (157+-9% of control, P<0.05). There was no significant change in

aquaporin-2 abundance, although there was a marked increase in aquaporin-3 abundance in spironolactone-treated rats (216+-28% of control, P<0.05). The results support the view that the regulation of ENaC by aldosterone is a consequence of mineralocorticoid receptor occupation.

2/7/34 (Item 33 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16075584 BIOSIS NO.: 200100247423
Changes in abundances of sodium transporters and sodium channel (ENaC) subunits in rat kidney in response to AT1 receptor blockade
AUTHOR: Beutler Kathleen T (Reprint); Nielsen Jakob (Reprint); Masilamani Shyama (Reprint); Brooks Heddwen L (Reprint); Knepper Mark A (Reprint)
AUTHOR ADDRESS: NHLBI, NIH, 10 Center Dr, MSC-1603, Bethesda, MD, 20892-1603, USA**USA
JOURNAL: FASEB Journal 15 (5): pA787 March 8, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Angiotensin II regulates renal sodium reabsorption both by direct effects on the renal tubule and via regulation of aldosterone secretion. Our previous studies have established that aldosterone increases the renal abundances of the thiazide-sensitive Na-Cl cotransporter (NCC) of the distal convoluted tubule and the alpha subunit of the amiloride-sensitive sodium channel (ENaC). To determine the long-term effect of angiotensin II on sodium transporter and sodium channel abundances along the renal tubule, we have tested the effect of administration of the AT1 receptor blocker candesartan by osmotic minipump (1 mg/kg/day s.c (n=6) vs. vehicle (n=6)) on the renal abundances of nine sodium transporter and channel proteins, and three aquaporins (AQPs) using semiquantitative %%%immunoblotting%%%. Dietary sodium intake was restricted to 0.5 meq/day in 200 g rats. (Lower dietary sodium intakes resulted in decreased glomerular filtration rate.) The plasma aldosterone concentrations were: control, 1.0+-0.3 nM; candesartan, 0.3+-0.1 nM; P=0.05. The candesartan infusion decreased the abundances of the proximal sodium-phosphate cotransporter (NaPi-2) to 53 +- 4 percent of control and the alpha subunit of ENaC to 53 +- 2 percent of control. In contrast, there were no significant decreases in the renal abundances of the proximal Na-H exchanger (NHE3), the proximal Na-bicarbonate cotransporter (NBC1), the bumetanide-sensitive Na-K-2Cl cotransporter (NKCC2), NCC, the beta or gamma subunit of ENaC, the alpha-1 subunit of Na-K-ATPase, AQP-1, AQP-2, or AQP-3. These results point to at least two sites of action of angiotensin II in regulation of transporter abundance, viz, the proximal tubule (downregulation of NaPi-2) and collecting duct (downregulation of alpha ENaC). The latter effect may be a result of reduced aldosterone levels.

2/7/35 (Item 34 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16074823 BIOSIS NO.: 200100246662
Hyperosmolarity induces a basolateral localization of the aquaporin-2 water channel
AUTHOR: van Balkom Bas W M (Reprint); van Raak Marcel; van Os Carel H (Reprint); van der Sluijs Peter; Deen Peter M T (Reprint)
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JOURNAL: FASEB Journal 15 (4): pA144 March 7, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In renal collecting ducts, vasopressin induces the redistribution of the Aquaporin-2 (AQP2) water channel from intracellular vesicles to the apical membrane. Drive by an osmotic gradient, water then enters the cell via AQP2 and exits the cell via basolaterally-located AQP3 and AQP4 and, consequently, urine is concentrated. Recent %%%immunohistochemical%%% data, however, revealed that in the cortical collecting ducts of water-deprived rats and in the inner medullary collecting ducts (IMCD) of control rats. AQP2 is also found in the basolateral membrane. Our goal was to identify the underlying mechanism for the basolateral localization of AQP2. Since potassium channels form heterotetramers and AQP2 is expressed as a tetramer, it was tested whether the formation of heterotetramers of AQP2 with AQP3 and/or AQP4 might direct AQP2 to the basolateral membrane. AQP-specific %%%immunoprecipitation%%% and %%%immunoblotting%%% of AQP2, AQP3 or AQP4 from oocytes expressing all three forms or from renal medullas or water-deprived rats revealed that neither AQP formed a heterotetramer with another AQP, although all were expressed as tetramers. Since the basolateral localization of AQP2 in the kidney is predominantly observed in conditions of increased osmolarity, it was tested whether hyperosmolarity could direct AQP2 to the basolateral membrane of wt10 cells, a stably-transfected Madin Darby Canine Kidney (MDCK) cell line in which AQP2 is normally routed to the apical membrane. Indeed, a 375 mOsm increase of the medium using NaCl, mannitol, sucrose or raffinose resulted in a basolateral and apical localization of AQP2. Dehydration of rats also elevates AQP2 expression, but analysis of different transfected MDCK clones revealed that with increased expression levels AQP2 was only localized in the apical membrane. Therefore, our data suggest that an increased osmolarity might cause the basolateral localization of AQP2 in (the IMCD of) the kidney.

2/7/36 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15605554 BIOSIS NO.: 200000323867

Dysregulation of renal aquaporins and Na-Cl cotransporter in CCl4-induced cirrhosis
AUTHOR: Fernandez-Llama Patricia; Jimenez Wladimiro; Bosch-Marce Marta; Arroyo Vicente; Nielsen Soren; Knepper Mark A (Reprint)
AUTHOR ADDRESS: National Institutes of Health, 10 Center Drive, Building 10, Room 6N260, Bethesda, MD, 20892-1603, USA**USA
JOURNAL: Kidney International 58 (1): p216-228 July, 2000 2000
MEDIUM: print
ISSN: 0085-2538
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Severe hepatic cirrhosis is associated with abnormal renal water retention. Methods: Semiquantitative %%%immunoblotting%%% was employed to investigate the abundance of the major renal aquaporins (water channels) and sodium-dependent cotransporters in kidneys from control rats and rats with cirrhosis secondary to chronic CCl4 inhalation. Results: The cirrhotic rats had ascites and manifested a water excretion defect detected by a standard water-loading test. The abundance of aquaporin-1 (the major aquaporin in the proximal tubule) was increased, an effect markedly accentuated in high-density membrane fractions prepared by differential centrifugation. Differential centrifugation studies demonstrated a redistribution of aquaporin-2 from high-density to low-density membranes, compatible with increased trafficking of aquaporin-2 to the plasma membrane. The abundance of aquaporin-3, but not aquaporin-2, was increased in collecting ducts of rats with CCl4-induced cirrhosis. The Na-K-2Cl cotransporter of the thick ascending limb showed no change in abundance. However, the abundance of the thiazide-sensitive Na-Cl cotransporter of the distal convoluted tubule was markedly suppressed in cirrhotic rats, possibly contributing to a defect in urinary dilution. Conclusions: In this model of cirrhosis, the development of a defect in urinary dilution may be multifactorial, with contributions from at least four abnormalities in transporter regulation: (1) an increase in the renal abundance of aquaporin-1, (2) a cellular redistribution of aquaporin-2 in the collecting duct compatible with trafficking to the plasma membrane without an increase in total cellular aquaporin-2, (3) an increase in the renal abundance of aquaporin-3, and (4) a decrease in the abundance of the thiazide-sensitive cotransporter of the distal convoluted tubule.

2/7/37 (Item 36 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15550698 BIOSIS NO.: 200000269011
Aquaporins in rabbit jejunal epithelium
AUTHOR: Wallace Laurie E (Reprint); Millar Grant A; Chung Brian M; O'Loughlin Ted V; Hardin James A; Gall Donald G
AUTHOR ADDRESS: Univ of Calgary, Calgary, AB, Canada**Canada
JOURNAL: Gastroenterology 118 (4 Suppl. 2 Part 2): pAGA A1140 April, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 101st Annual Meeting of the American Gastroenterological Association and the Digestive Disease Week. San Diego, California, USA May 21-24, 2000; 20000521

SPONSOR: American Gastroenterological Association
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

15109367 BIOSIS NO.: 199900369027
Developmental expression of ROMK in rat kidney
AUTHOR: Zolotnitskaya Anna; Satlin Lisa M (Reprint)
AUTHOR ADDRESS: Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, NY, 10029-6574, USA**USA
JOURNAL: American Journal of Physiology 276 (6 PART 2): pF825-F836 June, 1999 1999
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The apical secretory K+ (SK) channel in the principal cell represents the rate-limiting step for K+ secretion across the cortical collecting duct (CCD). Patch clamp analysis of maturing rabbit principal cells identifies an increase in number of conducting SK channels after the 2nd week of life (L. M. Satlin and L. G. Palmer. Am. J. Physiol. 272 (Renal Physiol. 41): F397-F404, 1997), apprx1 wk after an increase in activity of the amiloride-sensitive epithelial Na+ channel (ENaC) is detected. To correlate the postnatal increase in channel activity with developmental expression of ROMK, the molecular correlate of the SK channel, we used gene-specific probes to show a developmental increase in abundance of renal ROMK mRNA and a ROMK-specific antibody to examine the nephron distribution, localization, and abundance of this protein in developing rat kidney. Using antibodies directed against aquaporin-3 (AQP-3) and Tamm-Horsfall protein (THP), we confirmed that ROMK was expressed along theapical membranes of principal cells and thick ascending limbs of Henle (TALH) in adult kidney. Within the midcortex of the neonatal kidney, ROMK-positive segments revealed weak coincident staining with the TALH-specific antibody but did not colabel with an antibody directed against distal and connecting tubule (CNT)-specific kallikrein or the lectin Dolichos biflorus agglutinin (DBA), which labels proximal tubules and collecting ducts. In inner cortex and outer medulla of kidneys from 1-wk-old animals, ROMK protein was identified in medullary TALH (MTALH) and DBA-positive collecting ducts. By 3 wk of age, coincident ROMK and DBA expression was detected in midcortical and outer cortical CNTs and CCDs. %%%Immunoblot%%% analysis of plasma membrane-enriched fractions of maturing rat kidney revealed a developmental increase in a apprx40-kDa band, the expected size for ROMK. %%%Immunolocalization%%% of alpha-ENaC showed apical staining of a majority of cells in distal nephron segments after the 1st week ofpostnatal life. The beta- and gamma-ENaC subunit expression was routinely detected in a mostly cytoplasmic distribution immediately after birth, albeit in low abundance; gamma-ENaC showed some apical polarization. These results suggest that the postnatal increases in a

principal cell apical SK and Na+ channel activity are mediated, at least in part, by increases in abundance of ROMK message and protein and ENaC subunit proteins.

2/7/39 (Item 38 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15070216 BIOSIS NO.: 199900329876
Localization of aquaporin-3 mRNA and protein along the gastrointestinal tract of Wistar rats
AUTHOR: Ramirez-Lorca R; Vizuete M L; Venero J L; Revuelta M; Cano J; Ilundain A A; Echevarria M (Reprint)
AUTHOR ADDRESS: Departamento de Fisiologia y Biologia Animal, Facultad de Farmacia, Universidad de Sevilla, C) Tramontana s/n, E-41012, Sevilla, Spain**Spain
JOURNAL: Pfluegers Archiv European Journal of Physiology 438 (1): p94-100 June, 1999 1999
MEDIUM: print
ISSN: 0031-6768
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Since specific proteins responsible for water transport (aquaporins, AQPs) have been identified in a great variety of tissues, we decided to study the presence of AQP3 in the gastrointestinal tract (GIT) of Wistar rats. Poly(A+) RNA was purified from the mucosa of the stomach, jejunum, ileum and colon, and gross detection of AQP3 mRNA was done by Northern blot analysis. In situ hybridization studies were carried out to precisely localize the distribution of this transcript. Sections of the different tissues were hybridized with simeq400-bp (35S)riboprobes. The results presented here demonstrate that AQP3 is expressed throughout the GIT, with its expression in the colon and ileum greater than that in the stomach. %%%Immunohistochemistry%%% experiments, using a polyclonal antibody against AQP3, revealed that AQP3 protein is present at the basolateral membrane of the epithelial cells lining the villus tip of the small intestine and colon. The finding of AQP3 in the intestinal epithelia strongly suggests that this protein functions as a pathway for water transport in this epithelium.

2/7/40 (Item 39 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14740672 BIOSIS NO.: 199900000332
Regulation of Na-K-2Cl cotransporter abundance in rat thick ascending limb by vasopressin
AUTHOR: Ecelbarger C A (Reprint); Kim G-H; Mitchell C; Packer R K; Wade J B ; Knepper M A
AUTHOR ADDRESS: NHLBI, NIH, Bethesda, MD, USA**USA
JOURNAL: Journal of the American Society of Nephrology 9 (PROGRAM AND ABSTR. ISSUE): p18A Sept., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 31st Annual Meeting of the American Society of

Nephrology Philadelphia, Pennsylvania, USA October 25-28, 1998; 19981025
SPONSOR: American Society of Nephrology
ISSN: 1046-6673
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

2/7/41 (Item 40 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14518293 BIOSIS NO.: 199800312540
The perinatal expression of aquaporin-2 and aquaporin-3 in developing kidney
AUTHOR: Baum Michelle A; Ruddy Marcella K; Hosselet Christine A; Harris H William (Reprint)
AUTHOR ADDRESS: Renal Res. Lab., Div. Nephrol., Enders Build., Room 1260, Child. Hosp., 300 Longwood Avenue, Boston, MA 02115, USA**USA
JOURNAL: Pediatric Research 43 (6): p783-790 June, 1998 1998
MEDIUM: print
ISSN: 0031-3998
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The kidney provides an important contribution to permit the fetus to successfully transition to an independent existence by production of urine with significantly different osmolality compared with plasma. Although recent work has uncovered many aspects of the maturation and regulation of the renal concentrating and diluting mechanism, understanding of how alterations in the expression of aquaporin (AQP) water channels contribute to the formation of urine in the perinatal period is incomplete. Here, we report that both AQP-2 and -3 are expressed during fetal life as early as embryonic d 18 in ureteric buds of rat kidneys, where each is localized to the apical and basolateral membranes of epithelial cells, respectively. Northern analyses demonstrate that the 1.9-kb AQP-2 transcript is present in fetal and postnatal rat kidneys similar to that observed in adults. AQP-2 mRNA expression increases after d 3 of postnatal life. %%%Immunoblotting%%% reveals an increase in total kidney AQP-2 protein particularly with respect to its glycosylated form after postnatal d 3. AQP-3 protein also exhibits a similar alteration likely due to a similar increase in its glycosylation state. Both AQP-2 and AQP-3 display a distribution in the collecting ducts of human postnatal infants and adults identical to that exhibited in rat kidneys. These data show that both AQP-2 and -3 are present in collecting duct epithelia of fetal and postnatal kidneys. Thus, the reduced AVP-responsiveness and decreased urinary concentrating ability of the kidney during the fetal and immediate postnatal period does not appear to be caused by lack of AQP-2 or AQP-3 proteins.

2/7/42 (Item 41 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14428507 BIOSIS NO.: 199800222754

Evidence for the presence of aquaporin-3 in human red blood cells
AUTHOR: Roudier Nathalie; Verbavatz Jean-Marc; Maurel Christophe; Ripoche Pierre; Tacnet Frederique (Reprint)
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JOURNAL: Journal of Biological Chemistry 273 (14): p8407-8412 April 3, 1998 1998
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A facilitated diffusion for glycerol is present in human erythrocytes. Glycerol transporters identified to date belong to the Major Intrinsic Protein (MIP) family of integral membrane proteins, and one of them, aquaporin-3 (AQP3), has been characterized in mammals. Using an antibody raised against a peptide corresponding to the rat AQP3 carboxyl terminus, we examined the presence of AQP3 in normal and Colton-null (aquaporin-1 (AQP1)-deficient) human erythrocytes. Three %%%immunoreactive%%% bands were detected on %%%immunoblots%%% of both normal and Colton-null red cells, very similar to the bands revealed in rat kidney, a material in which AQP3 has been extensively studied. By %%%immunofluorescence%%%, anti-AQP3 antibodies stained the plasma membranes of both normal and Colton-null erythrocytes. Glycerol transport was measured on intact erythrocytes by stopped-flow light scattering and on one-step pink ghosts by a rapid filtration technique. Glycerol permeability values, similar in both cell types, suggest that AQP1 does not represent the major path for glycerol movement across red blood cell membranes. Furthermore, pharmacological studies showed that Colton-null red cells remain sensitive to water and glycerol flux inhibitors, supporting the idea that another proteinaceous path, probably AQP3, mediates most of the glycerol movements across red cell membranes and represents part of the residual water transport activity found in AQP1-deficient red cells.

2/7/43 (Item 42 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14349492 BIOSIS NO.: 199800143739
Fourfold reduction of water permeability in inner medullary collecting duct of aquaporin-4 knockout mice
AUTHOR: Chou C L; Ma Tonghui; Yang Baoxue; Knepper Mark A; Verkman A S (Reprint)
AUTHOR ADDRESS: 1246 Health Sci. East Tower, Cardiovascular Res. Inst., Univ. California San Francisco, San Francisco, CA 94143-0521, USA**USA
JOURNAL: American Journal of Physiology 274 (2 PART 1): pC549-C554 Feb., 1998 1998
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aquaporin (AQP)-3 and AQP4 water channels are expressed at the

basolateral membrane of mammalian collecting duct epithelium. To determine the contribution of AQP4 to water permeability in the initial inner medullary collecting duct (IMCD), osmotic water permeability (Pf) was compared in isolated perfused IMCD segments from wild-type and AQP4 knockout mice. The AQP4 knockout mice were previously found to have normal gross appearance, survival, growth, and kidney morphology and a mild urinary concentrating defect (T. Ma, B. Yang, A. Gillespie, E. J. Carlson, C. J. Epstein, and A. S. Verkman. J. Clin. Invest. 100: 957-962, 1997). Transepithelial Pf was measured in microdissected IMCDs after 18-48 h of water deprivation and in the presence of 0.1 nM arginine vasopressin (to make basolateral Pf rate limiting). Pf values (37degreeC; means +- SE in cm/s X 10-3) were 56.0 +- 8.5 for wild-type mice (n = 5) and 13.1 +- 3.7 for knockout mice (n = 6) (P < 0.001). Northern blot analysis of kidney showed that transcript expression of AQP1, AQP2, AQP3, and AQP6 were not affected by AQP4 deletion. %%%Immunoblot%%% analysis indicated no differences in protein expression of AQP1, AQP2, or AQP3, and %%%immunoperoxidase%%% showed no differences in staining patterns. Coexpression of AQP3 and AQP4 in Xenopus laevis oocytes showed additive water permeabilities, suggesting that AQP4 deletion does not affect AQP3 function. These results indicate that AQP4 is responsible for the majority of basolateral membrane water movement in IMCD but that its deletion is associated with a very mild defect in urinary concentrating ability.

2/7/44 (Item 43 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14236045 BIOSIS NO.: 199800030292
Aquaporins in complex tissues: I. Developmental patterns in respiratory and glandular tissues of rat
AUTHOR: King Landon S; Nielsen Soren; Agre Peter (Reprint)
AUTHOR ADDRESS: Dep. Biol. Chem., Johns Hopkins Univ. Sch. Med., 725 lN. Wolfe St., Baltimore, MD 21205-2185, USA**USA
JOURNAL: American Journal of Physiology 273 (5 PART 1): pC1541-C1548 Nov., 1997 1997
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Developmental expression of aquaporin water transport proteins is not well understood in respiratory tract or secretory glands; here we define aquaporin protein ontogeny in rat. Expression of aquaporin-3 (AQP3), AQP4, and AQP5 proteins occurs within 2 wk after birth, whereas AQP1 first appears before birth. In most tissues, aquaporin protein expression increases progressively, although transient high-level expression is noted in distal lung (AQP4 at postnatal day +2) and trachea (AQP5 at postnatal day +21 and AQP3 at postnatal day +42). In mature animals, AQP5 is abundant in distal lung and salivary glands, AQP3 and AQP4 are present in trachea, and AQP1 is present in all of these tissues except salivary glands. Surprisingly, all four aquaporin proteins are highly abundant in nasopharynx. Unlike AQP1, corticosteroids did not induce expression of AQP3, AQP4, or AQP5 in lung. Our results seemingly implicate aquaporins in proximal airway humidification, glandular

secretion, and perinatal clearance of fluid from distal airways. However, the studies underscore a need for detailed %%%immunohistochemical%%% characterizations and definitive functional studies.

2/7/45 (Item 44 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14233842 BIOSIS NO.: 199800028089
Aquaporins in complex tissues: II. Subcellular distribution in respiratory and glandular tissues of rat
AUTHOR: Nielsen Soren; King Landon S; Christensen Birgitte Monster; Agre Peter (Reprint)
AUTHOR ADDRESS: Dep. Biol. Chem., Johns Hopkins Univ. Sch. Med., 725 N. Wolfe St., Baltimore, MD 21205-2185, USA**USA
JOURNAL: American Journal of Physiology 273 (5 PART 1): pC1549-C1561 Nov., 1997 1997
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The molecular pathways for fluid transport in pulmonary, oral, and nasal tissues are still unresolved. Here we use %%%immunocytochemistry%%% and %%%immunoelectron%%% microscopy to define the sites of expression of four aquaporins in the respiratory tract and glandular epithelia, where they reside in distinct, nonoverlapping sites. Aquaporin-1 (AQP1) is present in apical and basolateral membranes of bronchial, tracheal, and nasopharyngeal vascular endothelium and fibroblasts. AQP5 is localized to the apical plasma membrane of type I pneumocytes and the apical plasma membranes of secretory epithelium in upper airway and salivary glands. In contrast, AQP3 is present in basal cells of tracheal and nasopharyngeal epithelium and is abundant in basolateral membranes of surface epithelial cells of nasal conchus. AQP4 resides in basolateral membranes of columnar cells of bronchial, tracheal, and nasopharyngeal epithelium; in nasal conchus AQP4 is restricted to basolateral membranes of a subset of intra- and subepithelial glands. These sites of expression suggest that transalveolar water movement, modulation of airway surface liquid, air humidification, and generation of nasopharyngeal secretions involve a coordinated network of aquaporin water channels.

2/7/46 (Item 45 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13877428 BIOSIS NO.: 199799511488
Expression and localization of the water channels in human autosomal dominant polycystic kidney disease
AUTHOR: Hayashi Matsuhiko (Reprint); Yamaji Yasuyoshi; Monkawa Toshiaki; Yoshida Tadashi; Tsuganezawa Hirohiko; Sasamura Hiroyuki; Kitajima Waichi ; Sasaki Sei; Ishibashi Kennichi; Maurmo Fumiaki; Saruta Takao
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JOURNAL: Nephron 75 (3): p321-326 1997 1997
ISSN: 0028-2766
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To characterize the cyst-lining cells in human autosomal dominant polycystic kidney disease (ADPKD), we performed %%%immunohistological%%% studies with specific antibodies against human aquaporin-2 (AQP-2, the vasopressin-regulated water channel) and aquaporin-3 (AQP-3), which are expressed only in collecting duct cells in the normal kidney. The polycystic kidney samples were obtained from 2 hemodialysis patient at uninephrectomy. %%%Immunohistochemical%%% studies revealed two types of staining of cyst-lining cells. Approximately 30% of all the cysts were simultaneously %%%immunostained%%% by both antibodies. Among these AQP-positive cysts, more than 90% of the cysts were intensely stained, with well-polarized localization of AQP-2 and AQP-3. In fewer than 10% of AQP-positive cysts, by contrast, %%%immunostaining%%% for AQP-2 and AQP3 was faint and no clearly polarized localization of the channels was observed. We examined the %%%immunostaining%%% in further detail by electron microscopy. Staining specific for AQP-2 was mainly observed in the apical membrane of cyst-lining cells. Moreover, staining specific for AQP-3 was observed in all of the AQP-2-positive cysts. It appeared unlikely that the variations in %%%immunostaining%%% observed under the light microscope had been induced by total disruption of water-channel polarity. The present study suggests that about 30% of the cysts in our cases of ADPKD were derived from the collecting duct cells and that the cyst-lining cells were well differentiated in terms of AQP expression.

13846410 BIOSIS NO.: 199799480470
%%%Immunolocalization%%% and effect of dehydration on AQP3, a basolateral water channel of kidney collecting ducts
AUTHOR: Ishibashi Kenichi (Reprint); Sasaki Sei; Fushimi Kiyohide; Yamamoto Tadashi; Kuwahara Michio; Marumo Fumiaki
AUTHOR ADDRESS: Second Dep. Intern. Med., Tokyo Med. Dental Univ., 1-5-45 Yushima, Bunkyo, Tokyo 113, Japan**Japan
JOURNAL: American Journal of Physiology 272 (2 PART 2): pF235-F241 1997 1997
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aquaporin-3 (AQP3) is unique in its structure (lowest homology with other aquaporins) and in its function (significantly conductive to both small nonelectrolytes and water). However, there is a controversy among researchers on its water transport and induction by dehydration. We examined its localization and the effect of dehydration on its expression in the kidney, as well as its water channel activity when expressed in Xenopus oocytes. In vitro translation using reticulocyte lysate revealed that the size of rat AQP3 was 26 kDa, and the band shifted to around 31 kDa with microsomal fraction, which was sensitive to the digestion with

N-glycosidase F. In Western blot analysis of rat kidney medulla, AQP3 appeared as a sharp band at 27 kDa and a broad band at 34-40 kDa. In %%%immunohistochemistry%%%, AQP3 was localized to principal cells and absent in intercalated cells in outer medulla. In inner medulla, AQP3 was restricted to inner medullary collecting duct (IMCD) cells. AQP3 was confined to the basolateral membrane of these cells. Although dehydration of rats for 2 days did not change the distribution pattern of AQP3 in IMCD cells, the dehydration increased AQP3 mRNA by twofold with slight increase of its protein level in kidney medulla. Finally, we confirmed its water channel activity when expressed in Xenopus oocytes. The human AQP3 stimulated osmotic water permeability by eightfold, which was inhibited by 0.3 mM mercury chloride by 34% and reversed by beta-mercaptoethanol. Our results indicate that AQP3 is a glycosylated protein and a mercury-sensitive water channel localized at the basolateral membrane of principal cells and IMCD cells, and its expression is induced by dehydration at both protein and mRNA level.

2/7/48 (Item 47 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13846407 BIOSIS NO.: 199799480467
Expression of AQP family in rat kidneys during development and maturation
AUTHOR: Yamamoto Tadashi (Reprint); Sasaki Sei; Fushimi Kiyohide; Ishibashi Kenichi; Yaoita Eishin; Kawasaki Katsutoshi; Fujinaka Hidehiko; Marumo Fumiaki; Kihara Itaru
AUTHOR ADDRESS: Dep. Pathol., Inst. Nephrol., Niigata Univ. Sch. Med., 1-757 Asahimachi-dori, Niigata 951, Japan**Japan
JOURNAL: American Journal of Physiology 272 (2 PART 2): pF198-F204 1997
1997
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The mRNA expression and localization of the aquaporin (AQP) family in rat kidney were examined by ribonuclease protection assay and %%%immunohistochemistry%%%. AQP1, AQP2, AQP3, and AQP4 mRNA were hardly detectable in 16-day gestation fetuses. AQP1 mRNA was explosively expressed at 1 wk, keeping the level throughout life. AQP2 mRNA expression was apparently noticed in 18-day fetuses and was enhanced gradually with age to reach a plateau at 4 wk. AQP3 and AQP4 mRNA expression was significantly found at birth but was not changed remarkably thereafter. AQP2 protein appeared first at the apical side of collecting duct cells in 18-day fetuses. The staining intensity at the site increased with age, and basolateral staining was added in adult rats. AQP3 was distinctly demonstrated at the basolateral side of collecting duct cells after birth, and the staining intensity was almost stable throughout life. The progressive induction of AQP2 expression in the first 4 wk after birth is presumed to contribute to the maturation of urinary concentrating capacity during the kidney development.

2/7/49 (Item 48 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13783435 BIOSIS NO.: 199799417495
Reduced renal medullary water channel expression in puromycin aminonucleoside-induced nephrotic syndrome
AUTHOR: Apostol Emmanuel; Ecelbarger Carolyn A; Terris James; Bradford Ako D; Andrews Peter; Knepper Mark A (Reprint)
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JOURNAL: Journal of the American Society of Nephrology 8 (1): p15-24 1997 1997
ISSN: 1046-6673
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The aquaporins are molecular water channels that mediate transcellular water transport across water-permeable epithelia. To investigate the cause of the concentrating defect in the nephrotic syndrome, %%%immunoblotting%%% using membrane fractions from inner medulla was utilized to assess the level of expression of four aquaporin water channels in vehicle-treated versus puromycin aminonucleoside (PAN)-treated rats. Scanning electron microscopy demonstrating loss of glomerular foot processes and measurements of urinary protein excretion confirmed the efficacy of the PAN treatment. In rats receiving PAN, there was an increase in plasma vasopressin, without a change in plasma sodium concentration. Inner medullary tissue hypertonicity was sustained in PAN-treated rats while the urinary osmolality was low, pointing to defective osmotic equilibration across the collecting ducts in PAN-nephrosis. Among collecting duct aquaporins, there was an 87% decrease in aquaporin-2 expression and a 70% decrease in aquaporin-3 expression in the inner medulla, whereas aquaporin-4 expression was unaltered. Transmission electron microscopy of the inner medullary collecting ducts of PAN-treated rats showed normal-appearing cells. Thus, PAN-nephrosis is associated with an extensive downregulation of collecting duct water channel expression despite increased circulating vasopressin, providing an explanation for the concentrating defect associated with the nephrotic syndrome.

2/7/50 (Item 49 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13574180 BIOSIS NO.: 199699208240
Long-term regulation of four renal aquaporins in rats
AUTHOR: Terris James; Ecelbarger Carolyn A; Nielsen Soren; Knepper Mark A (Reprint)
AUTHOR ADDRESS: Natl. Inst. Health, Build. 10, Room 6N307, 10 Center Drive MSC 1598, Bethesda, MD 20892-1598, USA**USA
JOURNAL: American Journal of Physiology 271 (2 PART 2): pF414-F422 1996 1996
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The aquaporins are molecular water channels expressed in the

kidney and other organs. To investigate long-term regulation of renal expression of these water channels, we carried out %%%immunoblotting%%% studies using membrane fractions from rat renal cortex and medulla. Both 48-h water restriction in Sprague-Dawley rats and 5-day arginine vasopressin (AVP) infusion in Brattleboro rats caused significant increases in the expression levels of two aquaporins, aquaporin-2 and aquaporin-3, while the levels of aquaporin-1 and aquaporin-4 were unchanged. The increases in aquaporin-2 and aquaporin-3 expression were seen in inner and outer medulla as well as cortex. Ablation of the corticomedullary interstitial osmotic gradient with an infusion of furosemide did not eliminate the upregulatory response to AVP infusion in Brattleboro rats. Furthermore, 5-day furosemide infusion to Sprague-Dawley rats did not decrease expression levels of the collecting duct aquaporins, but rather increased them. We conclude that the expression of aquaporin-2 and aquaporin-3, but not aquaporin-1 or aquaporin-4, is increased in response to elevated circulating AVP. Because regulation of aquaporin-2 and aquaporin-3 levels was observed in the cortex and because osmotic gradient ablation did not abrogate the increase, we conclude that changes in interstitial osmolality are not necessary for the AVP-induced upregulation of aquaporin-2 and aquaporin-3 expression.

2/7/51 (Item 50 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13462924 BIOSIS NO.: 199699096984
Changes in aquaporin-2 protein contribute to the urine concentrating defect in rats fed a low-protein diet
AUTHOR: Sands Jeff M (Reprint); Naruse Masahiro; Jacobs Joely D; Wilcox Josiah N; Klein Janet D
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JOURNAL: Journal of Clinical Investigation 97 (12): p2807-2814 1996 1996
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Low-protein diets cause a urinary concentrating defect in rats and humans. Previously, we showed that feeding rats a low (8%) protein diet induces a change in urea transport in initial inner medullary collecting ducts (IMCDs) which could contribute to the concentrating defect. Now, we test whether decreased osmotic water permeability (P-f) contributes to the concentrating defect by measuring P-f in perfused initial and terminal IMCDs from rats fed 18 or 8% protein for 2 wk. In terminal IMCDs, arginine vasopressin (AVP)stimulated osmotic water permeability was significantly reduced in rats fed 8% protein compared to rats fed 18% protein. In initial IMCDs, AVP-stimulated osmotic water permeability was unaffected by dietary protein. Thus, AVP-stimulated osmotic water permeability is significantly reduced in terminal IMCDs but not in initial IMCDs. Next, we determined if the amount of %%%immunoreactive%%% aquaporin-2 (AQP2, the AVP-regulated water channel) or AQP3 protein was altered. Protein was isolated from base or tip regions of rat inner medulla and Western analysis performed using polyclonal antibodies to rat AQP2 or AQP3 (courtesy of Dr. M.A. Knepper,

National Institutes of Health, Bethesda, MD). In rats fed 8% protein (compared to rats fed 18% protein): (a) AQP2 decreases significantly in both membrane and vesicle fractions from the tip; (b) AQP2 is unchanged in the base; and (c) AQP3 is unchanged. Together, the results suggest that the decrease in AVP-stimulated osmotic water permeability results, at least in part, in the decrease in AQP2 protein. We conclude that water reabsorption, like urea reabsorption, responds to dietary protein restriction in a manner that would limit urine concentrating capacity.

0007982957 SUPPLIER NUMBER: 2009141813
Osmosensitivity of prolactin cells is enhanced by the water channel aquaporin-3 in a euryhaline Mozambique tilapia (Oreochromis mossambicus)
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Journal: American Journal of Physiology - Regulatory Integrative and Comparative Physiology (Am. J. Physiol. Regul. Integr. Comp. Physiol.), v296, n2, (R446-R453), 2009, United States
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URL: http://ajpregu.physiology.org/cgi/reprint/296/2/R446
PUBLISHER: American Physiological Society
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DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 44

In teleost fish, prolactin (PRL) has important actions in the regulation of salt and water balances in freshwater (FW) fish. Consistent with this role, the release of PRL from the pituitary of the Mozambique tilapia is stimulated as extracellular osmolality is reduced. Stretch-activated calcium-permeant ion channels appear to be responsible for the initiation of the signal transduction that leads to increased PRL release when PRL cells are exposed to reductions in extracellular osmolality. In this study, we examined a possible involvement of the aquaporin-3 (AQP3) water channel in this osmoreceptive mechanism in PRL cells of the tilapia. AQP3 expression levels in the rostral pars distalis of the pituitary, consisting predominantly of PRL cells, were higher in fish adapted to FW than in seawater (SW)-adapted fish. %%%Immunohistochemical%%% studies revealed that AQP3 is located in the cell membrane and perinuclear region of PRL cells, with more intense %%%immunosignals%%% in PRL cells of FW-adapted fish than in those of SW fish. In FW PRL cells, the magnitude of hyposmoticity-induced cell volume increase was greater than that seen in SW PRL cells. Mercury, a potent inhibitor of AQP3, inhibited hyposmoticity-induced cell volume increase and PRL release from FW PRL cells. The inhibitory effect of mercury was partially restored by beta-mercaptoethanol, whereas no effect of mercury was observed on PRL

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release stimulated by a depolarizing concentration of KCl, which induces Ca
SUP 2+ influx and stimulates the subsequent Ca SUP 2+ -signaling pathway.
These results indicate significant contribution of AQP3 to osmoreception in
PRL cells in FW-adapted tilapia. Copyright (c) 2009 the American
Physiological Society.
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       18jun09 16:33:08 User219511 Session D765.4
            $1.83    0.519 DialUnits File154
               $0.24  1 Type(s) in Format  7
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            $2.60  1 Types
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     $0.26  TELNET
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File 411:DIALINDEX(R)

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   You have 27 files in your file list.
   (To see banners, use SHOW FILES command)
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Your SELECT statement is:
   s pro96271

          Items   File
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? bye
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Logoff: level 05.24.00 D  16:33:20
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